

BRAIN GLYCOGEN METABOLISM DURING HYPOGLYCEMIA: ROLE IN HYPOGLYCEMIA ASSOCIATED AUTONOMIC FAILURE, MEMORY, AND NEURONAL CELL DEATH

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We hypothesize that brain glycogen, a stored form of glucose, may provide fuel for the brain conferring both negative and positive effects throughout the brain. The over accumulation of brain glycogen, or supercompensation, is hypothesized to exacerbate hypoglycemia associated autonomic failure (HAAF), promote memory and learning, and reduce neuron cell death during severe episodes of hypoglycemia. It was determined that brain glycogen supercompensation does occur in the mouse 6 hours following single and recurrent hypoglycemic episodes, but it is not likely a significant mechanism behind HAAF due to the supercompensation subsiding at 27 hours following the hypoglycemic episodes. In regard to memory and learning, brain glycogen is not required for motor skill learning while euglycemic, however, it does enhance motor memory while hypoglycemic as determined using a rotarod treadmill in mouse. In regard to associative learning, brain glycogen is important for contextual, but not cued, memories while both euglycemic and hypoglycemic, as assessed by contextual and cued fear conditioning. Two different genetically engineered models of mice lacking brain glycogen yielded opposing results when assessing whether brain glycogen is neuroprotective during severe and prolonged hypoglycemia. In conclusion, brain glycogen does not appear to play a role in HAAF, is important for learning and memory, and its role in neuronal cell survival during hypoglycemia requires further study.